MATTERS ARISING

Teenagers and the risk of STD

The recent paper on teenagers and the risk of sexually transmitted diseases by Mellanby *et al* demonstrated that in a cross section of 15–16 year olds knowledge of STD was poor.[1] Only 28% of answers to five questions related to STD were correct. We thought it would be of interest to include the same five questions in a questionnaire administered to new University students attending the recent "Freshers Fayre" at University College, London. Students passing our "Sexual Health" stall were asked to complete the questionnaire. Few students declined but clearly the students were not a randomly selected sample.

There were 529 completed questionnaires. Greater knowledge was evident in this population with 60% of answers to the five questions being correct. When compared with the teenager study a higher proportion of correct responses was obtained for all five questions. In the teenager study the questions most frequently answered incorrectly were first, a belief that condoms give total protection against STD (56% incorrect), and second that HIV is now the commonest sexually transmitted disease (53% incorrect). In the students we questioned the proportion of incorrect responses to these two questions was considerably lower at 30% and 20% respectively.

The table lists the answers obtained in our survey using the five questions used by Mellanby *et al* plus four additional questions. Although the students' knowledge of STD and sexual health awareness was generally good almost half those questioned felt that they had put themselves at risk of an STD. Perhaps this reflects the crucial gap between knowledge and practice of safer sex.

We found that providing information at a "Freshers Fayre" stall was a productive and simple way of promoting sexual health and increasing awareness of the services offered by this genitourinary medicine clinic. Some 1500 students obtained information leaflets from us over two days and the questionnaire provided a focus for discussion. We would encourage other genitourinary medicine clinics serving student populations to look for similar opportunities to disseminate knowledge and increase awareness of sexually transmitted infections and sexual health.

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1 Mellanby A, Phelps F, Lawrence C, Tripp JH. Teenagers and the risks of sexually transmitted diseases; a need for the provision of balanced information. *Genitourin Med* 1992; **68**:241–4.

Table Answers to questionnaire (n = 529)

	True	*False*	*Don't know*
1 HIV is the only sexually transmitted disease that cannot be cured.	32%	58%	10%
2 HIV is now the most common sexually transmitted disease.	20%	65%	15%
3 You can catch warts from sexual intercourse.	72%	12%	16%
4 Using a condom during sexual intercourse will give you total protection against sexually transmitted diseases.	30%	66%	4%
5 You can catch chlamydia from sexual intercourse.	40%	6%	54%
	Yes	*No*	*Don't know*
6 Does vaseline/baby oil/oils break male condoms?	59%	22%	19%
7 Should you have a condom on at the moment you penetrate?	94%	3%	3%
8 If you've got a spot or sore on your genitals does this increase your risk of HIV when having sex?	71%	13%	16%
9 Do you feel that you have ever put yourself at risk of a sexually transmitted infection?	47%	47%	6%

A colposcopic case control study of cervical squamous epithelial lesions

We are concerned that the findings of Dr Evans and his colleagues presented in their colposcopic study of genitourinary medicine clinic attenders[1] do not support their major conclusions that anogenital warts are not a risk factor for histological cervical HPV or CIN.

The first methodological difficulty with the study is the assumption that routine histopathological reporting can reliably diagnose the features of cervical HPV infection. Robertson *et al*[2] confirmed previous observations of significant variability in histopathological reporting of cervical biopsies. They provided data concerning the reliability and reporting features of HPV infection alone and showed a Kappa statistic of 0·11, indicating very poor reproducibility.

A more fundamental problem lies in the way the study results have been analysed. The authors report that they conducted a case control study in order to discover whether anogenital warts were an indication for colposcopy, because of their possible role as a marker for cervical intra-epithelial neoplasia (CIN) or human papillomavirus infection (HPVI).

Two criteria were used for proceeding to colposcopy in the study, namely, a history of, or current, anogenital warts, or an abnormal smear. The authors find (as have many before) that there was a strong association between abnormal smear and CIN. The association between anogenital warts and CIN was less strong. It is therefore argued that anogenital warts are relatively protective for CIN.

For a case control comparison to provide a valid estimate of the relative risk associated with an exposure, both cases and controls must be representative of all those with similar respective disease status in the population under consideration. In particular, inclusion in the study must be independent of the exposure under consideration to avoid selection bias. This latter condition is systematically violated in the author's study, since those women without warts must have abnormal cytology, which is known to be associated with CIN and HPVI.

A simple hypothetical example will illustrate the problem. Suppose we choose to study 100 women. Fifty of these are included because they have dyskariotic smear and no warts, and the other 50 because they have warts and no dyskariosis. Suppose the risk of CIN among women with dyskariosis to be 0·4 (RR = 16) and that among women with warts to be 0·1 (RR = 4), that is assuming that the risk for women with neither is 0·025. By applying these risks to our hypothetical sample we obtain the numbers of subjects in each group who have CIN.

Dyskariosis and CIN = 50 × 0·4 = 20
Dyskariosis but no CIN = 50 × 0·6 = 30
Warts and CIN = 50 × 0·1 = 5
Warts but no CIN = 50 × 0·9 = 45

We now perform a case-control comparison in the manner of the authors:

	No CIN	CIN	
Warts	45/75	5/25	(OR = 0·166)

Thus warts appear to be negatively correlated with CIN despite a true relative risk of 4.

It would appear that the negative association between anogenital warts and CIN reported in the authors' study suggests merely that women who have warts are less likely to have CIN than women with dyskariosis. The conclusions of the study are therefore entirely unsupported. Furthermore the authors themselves note that 16% of those women found to have warts on examination were found to have CIN, and 18% of women with a past history of warts had CIN. A reanalysis of these valuable data based on a clearer understanding of epidemiological methods and principles should prove of considerable interest.

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1 Evans BA, Bond RA, MacRae KD. A colposcopic case-control study of cervical squamous intraepithelial lesions in women with anogenital warts. *Genitourin Med* 1992;**68**: 300–4.
2 Robertson AJ, Anderson JM, Swanson Beck JS, *et al*. Observer variability in histopathological reporting of cervical biopsy specimens. *J Clin Pathol* 1989;42:231–8.

Evans et al reply:

Histological features of HPVI have been shown to correlate well with HPV detection by DNA-DNA hybridisation.[1] It would appear that the Scottish pathologists lacked experience in recognising these features.[2] Our finding of significant associations with cervical HPVI is further evidence that our data are meaningful and not random.

Unfortunately, Dr Renton and his colleagues have misunderstood our study and postulate a hypothetical example that is misleading. Put simply, the influence of warts, if any, on CIN in dyskaryotic patients is based on a comparison between patients with dyskaryosis alone and patients who

have both dyskaryosis and warts. A group with both dyskaryosis and warts is not included in the hypothetical example. If it had been, assuming that dyskaryosis and warts were independent risk factors for CIN, the relative risk for the group with both dyskaryosis and warts would be $16 \times 4 = 64$. This would imply that 100% of the group with both dyskaryosis and warts would have CIN. The 2×2 table would now be:-

	CIN	No CIN
Warts	55	45
No Warts	20	30

giving a relative risk of 1·83—more than 10 times the 0·17 obtained by Dr Renton and his colleagues from their inappropriate example.

We thank your correspondents for their combined interest in our paper.

1 Jenkins D, Tay SK, McCance DJ, *et al.* Histological and immunocytochemical study of cervical intraepithelial neoplasia (CIN) with associated HPV6 and HPV16 infections. *J Clin Pathol* 1986;**39**:1177–80.
2 Robertson AJ, Anderson JM, Swanson BECH, *et al.* Observer variability in histopathological reporting of cervical biopsy specimens. *J Clin Pathol* 1989;**42**:231–8.

National STD trends in Zambia 1987-89

In their review of national STD trends in Zambia, Matondo concludes that there has been a decline in STDs since 1987.[1] This, it is stated, has occurred in spite of the increase in the number of STD clinics.

I wish to point out, however, that the data presented do not make a strong case for reaching this conclusion. The figures presented are absolute numbers of total cases and number of cases of each particular condition. This does not clearly show any shift in any direction as the data are influenced by various qualitative and quantitative factors. The assertion that because all clinics reported a decline suggests a genuine decline in STDs is not valid and needs clarification. This assertion can only be proved by establishing that a similar decline in outpatient attendances did not occur. It is known, for instance, that the economic situation in Zambia worsened during the period under study. This resulted, as expected, in a reduction in the resources allocated for health services. Consequently, there was a reduction in the availability of drugs and personnel with a subsequent decrease in hospital utilisation in some years. Health facility attendance for all conditions declined over the years though not uniformly. Although a decline in numbers of STD attendances did occur during the period in question, there was no decline if taken as a proportion of the total number of attendances. The decline reported is therefore an apparent decline and not a genuine one.

Individuals who do not seek medical care from health centres and hospitals go elsewhere, as Dr. Matondo rightly points out. It is necessary, therefore to study STD trends among private practitioners, traditional healers and other informal health workers before being conclusive on declines. It is possible that there is an inverse relationship between the number of people who attend hospitals and those seek treatment elsewhere.

Although the AIDS control programme in Zambia was established in 1986 and the STD programme in 1980, it was not until 1988 that massive health education campaigns were initiated. It is not likely that there were positive outcomes of these initiatives as early as 1988. As STD trends can be used as a surrogate marker for sexual behaviour change, trends in HIV infection also can be used as a proxy indicator of STD trends. Contrary to Matondo's findings, HIV surveillance data have shown increases in sero-prevalence rates in antenatal clinic attenders and blood donors during that period.[2]

In a paper presented at the International Conference on AIDS in Africa, Kinshasa, we reviewed data from STDs in Zambia and were able to conclude that it was possible to infer wrongly that a decline had occurred if the data did not take account of various quantitative and qualitative factors.[3] In addition we showed that when rates are calculated using out-patient attendances as denominators fluctuations, and not a steady decline in trends, is noted.

It is for this reason that the data presented should have considered rates and not absolute numbers. In calculating the rates, the total number of individuals attending a particular site for any reason should be taken as a denominator. This is so that the biases introduced by factors that affect hospital services utilisation and provision are considered.

I wish to suggest, therefore, that Matondo re-analyse his data and use out-patient attendance for each period as a denominator. This will allow comparisons of rates as opposed to absolute numbers. In addition we suggest that tests of significance are performed on the data to validate whether any changes in trends are statistically significant and present a genuine decline in STD trends.

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1 Matondo P. National STD trends in Zambia: 1987–89 *Genitourin Med* 1992;**68**:192–8
2 Tembo G. Hira S. *Epidemiological review of HIV infection in Zambia.* V International Conference on AIDS, 1989, Montreal, Canada.
3 Tembo G Hira, S, Van Praag E. *Monitoring STD trends: quantitative and qualitative issues.* V International conference on AIDS in Africa, Kinshasa, 1990.

HIV infection in Tirupathi, India

I refer to the comments of Ravi Sockanathan, *Genitourin Med*, 1992;**68**:199. I must point out that vast numbers of non-Hindus also exist in Tirupathi and these include Muslims, Christians and others. It is therefore completely inappropriate to single out Hindus. Also, in a country with so much illiteracy as India, posters on sexual topics would be ill received. Television might help but what is really needed is education of the population as a whole. In the field of genitourinary medicine in particular much attention needs to be paid to differences between cultures.

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Sockanathan replies:

I refer to the comments of Dr S Tharakaram and value his suggestions, indicating that TV programmes and education of the population as a whole would raise the public awareness of the prevalence of HIV infection in Tirupathi and South India. However, he has failed to appreciate that holiday makers from Europe who are unaware of the prevalence of the disease in these areas, will certainly not be influenced by health education campaigns. Radio and television programmes may be a source of information for the local population only. Therefore the lay press published in the western world indicating the prevalence of the infection in these areas will certainly increase the awareness amongst holiday makers and some pilgrims!

Illiteracy in India, should not form a barrier for health education programmes. A standard protocol for the prevention and control of sexually transmitted diseases[1] should be followed in the tropics and developing countries as recommended by the World Health Organisation.

Although the epidemiology health education programmes of sexually transmitted diseases in the tropics vary from that in the developing countries,[2] it is well known that British yardsticks are commonly used in many parts of the world including India.

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1 Bennett FJ. Control of sexually transmitted diseases in the tropics and developing countries. In "*Sexually Transmitted Diseases in the Tropics*" Osoba AO, (ed). Bailliere; London. 1987.
2 Wilcox RR. VD education in developing countries. A comparison with developed countries. *Br J Venereal Dis* 1976;**52**:88.

Exophytic cervical warts—an indication for colposcopy?

In their recent paper, Evans *et al* conclude that external anogenital warts are not a risk for subclinical cervical HPV infection or for CIN and therefore not an indication for colposcopy.[1] In contrast to external exophytic warts, cytological evidence of cervical HPV infection was strongly associated with all grades of CIN. The authors suggest that external exophytic anogenital warts may in fact have a protective effect on the genesis of CIN. However, Evans *et al* make no comment on the relationship of exophytic cervical warts, as distinct from subclinical cervical HPV infection, to CIN.

Approximately 6% of women with genital warts may have exophytic cervical warts.[2] The practice at this unit is to perform colposcopy on all women with clinically apparent exophytic cervical warts regardless of the result of cervical cytology. In a preliminary study we reviewed the cytological, colposcopic and histological findings of all patients who had colposcopy performed primarily for this indication. Thirty four patients were identified over a 6 month period, of whom 82·4% had concomitant vulval warts. Only four (11·7%) women had cytological evidence of HPV infection. Dyskaryosis was found in 9/34 (26·5%). Twelve patients had normal cytology, two of whom had CIN (grade 2) on histology (negative predictive value 83·3%). Nineteen patients had low grade smears (inflammatory/borderline, wart virus infection, mild dyskaryosis) of whom four had CIN 1 and two CIN 2. Overall 9/34 (26·5%) women